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ORTHOSTATIC HYPOTENSION

J. J. Grimm

Translation of "L'hypotension orthostatique," Revue suisse de médecine (Praxis), 69, No. 16, 518-521, 1980

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16. Abstract

Following a brief physiopathological review, the author classifies orthostatic hypotension into three groups: organic, functional and medication-dependent. He stresses the importance of etiological diagnosis, the use of objective tests, and appropriate therapy, especially concerning the organic forms.

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ORTHOSTATIC HYPOTENSION

J. J. Grimm
University Medical Policlinic, Lausanne

I. Introduction

Orthostatic hypotension or the orthostatic syndrome is characterized by a symptomatology related to a drop in arterial pressure. Its clinical expression is the result of a deficiency or lack of coordination of regulatory mechanisms assuring a cerebral blood flow sufficient for a standing position. By definition it occurs during the change from clinostatism to orthostatism. /1*

Veyrat (1972) has defined the symptoms of orthostatic hypotension as the following: "the patient complains of vision bothered by the appearance of black spots in the visual field, occasional headaches, ringing in the ears and palpitations; he feels tired and dizzy. This weakness can end in an orthostatic collapse or a syncope with loss of consciousness. Objectively one observes paleness, excessive sweating, tachycardia and a drop in arterial pressure. With the return to a clinostatic state all of these symptoms disappear rapidly." It is important to distinguish orthostatic hypotension from another illness, a vagal illness (common faint). More frequent than the orthostatic syndrome, the vagal illness is also characterized by an inadequate response of the vegetative nervous system in which one notes a cholinergic predominance: excessive sweating, increase in peristaltic activity of the digestive tract, nausea and bradycardia. It is interesting to record the variation in arterial pressure and consequentially, the symptomatology from one day to another in conditions imposed on the same patient.

II. Physiopathology of the orthostatic syndrome /2

The set of symptomatology can be reduced to certain hemodynamic problems. These latter are always tributaries of the neurologic and

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* Numbers in the margin indicate pagination in the foreign text.

humoral phenomena whose study can be determinant during research of an etiology.

The primum novens is an accumulation of blood in the inclined regions and, consequently, a decrease in the cardiac venous return. The drop in cardiac flow involves a drop in arterial pressure. Inhibition of activity of the carotid and aorta baroreceptors follows a neural vegetative stimulation, meaning, in particular, an increase in cardiac frequency, a vasoconstriction of the arteriole and the capacitance of the vessels. Stimulation of the sympathoadrenergic axis involves a secretion of catecholamines and much later of renin. A deficiency or a dysfunction of one of the regulatory mechanisms can involve an orthostatic syndrome.

III. Etiology of Orthostatic Hypotension

One can divide the etiologies of orthostatic hypotension into three groups.

a) Consecutive orthostatic hypotension with an organic injury to the autonomous nervous system (see table) or of the cardiocirculatory apparatus. This involves organic orthostatism.

b) Functional orthostatic hypotension is like many physiological conditions occurring in pregnancy, the state of vagotonia at the end of the night or after prolonged bed rest. It is also classed among infectious diseases, febrile states of all origins and finally the entire situation where the vascular channel is abnormally large in relation to the volume circulating. Certain authors rank the orthostatic syndrome as constitutional, particularly affecting subjects of the leptosome type. All orthostatic hypotensions are spontaneously reversible; in general, this is the case for those related to the affections enumerated in a).

c) Medication-dependent orthostatic hypotension: diuretic, antihypertension, antidepressant, etc....

IV. Tests of the Neurovegetative Function

Certain tests proposed evidently do not dispense with an anamnesis particularly carefully directed at the neurovegetative functions. A good test must be simple, acceptable to the patient, viable and sensitive. None of the tests proposed is specific to a single etiology. It is important to repeat them.

a) Orthostatism test

The most widespread form is the Schellong test which is a strength test. This latter is poorly quantifiable and difficult to interpret. It comes from the department of orthostatic hypotension. Most of the authors propose only the curves of tension response and the tension response of "typical" pulses, seldom defined in a quantative manner.

We propose measurements of arterial pressure and pulse at the following rate: 3 values after at least 15 minutes of clinostatism, then 1 measurement every minute for the first 5 minutes of orthostatism, and finally at 10 and 15 minutes. If the pressure has dropped between the 10th and the 15th minute, we suggest a final measurement at 20 minutes. We prefer, whenever possible, a change in the position made by the patient himself rather than changing the position of the rotating table; this corresponds to a more physiological situation. The patient must not always be supported or remain immobile for the entire length of the test.

The drop in maximum physiological pressure is about 30 mm Hg for the systolic pressure according to certain authors. There are others with whom we have had a satisfactory experiment and a good correlation with the clinical, using a mean arterial pressure (PAM = diastolic pressure + 1/3 differential pressure). A drop of PAM of more than 10 mm Hg is pathologic. The limits are obviously arbitrary.

Here, finally, are two other tests which make it possible to confirm a suspicion of dysfunction in the vegetative nervous system.

b) Variations in cardiac frequency during the Valsalva test

The test is characterized by an acute increase in intrathoracic pressure obtained by contraction of the expiratory muscles against an obstacle, the glottis closed or finally a manometer. The subject is put in a semi-setting position with the legs flat. The manometer is attached to the mouth by a flexible tube and a mouthpiece. When he is at rest and breathing normally, the patient abruptly contracts the expiratory muscles until a pressure of 30 mm Hg is obtained without deep preliminary inspiration. He maintains this pressure for 12 seconds. Instead of following the development of arterial pressure as in the traditional test, one records an ECG at 25 mm/s during a period of 30 seconds after the maneuver. The development of cardiac frequency is characterized by a tachycardia preceding the end of the maneuver and a bradycardia after the latter. The tachycardia is caused by a vagal inhibition induced by the drop in arterial pressure. Whereas the bradycardia is the result of an increase in arterial pressure following the end of the test (rebound). Many authors, including Levin, have defined an index, the quotient of the RR interval of the ECG which is the longest, according to Valsalva and the RR interval which is the shortest during the maneuver. <u>This ratio must be larger than 1.2 or 1.5</u> according to the authors of [3].

c) Variations of cardiac frequency during the orthostatism test (see figure)

An acceleration of cardiac frequency during the change from clinostatism to orthostatism in a normal individual has been known for a long time. Short-term variations in cardiac frequency (during the first minute) on the contrary, have been described only recently [4].

The patient remains lying down until cardiac frequency has been stabilized. Then he gets up suddenly, in no more than 5 seconds, and remains standing without support for one minute. An ECG is recorded at a rate of 50 mm/s starting with the last seconds of clinostatism and lasting for the first minute of orthostatism. In a healthy subject

one observes a maximun cardiac acceleration by the 15th beat after changing position and a relative bradycardia at the 30th beat. Ewing and collaborators have defined the ratio of RR intervals of the ECG (30th/15th) as being 1.2 in the average subject and lower than or equal to 1 in patients having damage to the autonomous nervous system.

d) Interpretation

The results of the two latter tests are characterized by a decrease in values in the case of damage to the automomous nervous system. On the other hand, one encounters a whole series of curiosities, deficiencies, but also functional and neurovegetative dystonic hyperreactivity. The orthostatism test does not make it possible to distinguish the organic forms from the functional. A pathologic result in the three tests is that each time in the sense of attenuation of autonomous responses there is always a suggestion of an organic deficiency. In practice, one must be aware of the great frequency of orthostatic hypotensions which are called functional and medication-dependent. These forms require treatment much less often if there is not a primary affection or suppression by the responsible medication. When the practitioner suspects an organic form we propose the following measures:

- exclusion of cardiac or vascular affection, for example, an important varicose status;

- in a case where deficiency is suspected in the autonomous nervous system this must be confirmed by the tests described above.

The study of a precise etiology is often then the province of a specialist, in particular a neurologist.

V. Therapeutic

It is important to insist on precision in etiology diagnosis on which the choice of treatment will depend in part.

a) In the case of functional orthostatic hypotension or that related to the taking of medication, one should suppress the agent or the etiologic affection.
b) For organic forms affecting the vegetative nervous system (we will not deal with treatment of diseases of cardiovascular orgin in the strict sense) we propose the following measures:

1. Physical means. Depending on the type of orthostatic hypotension, a supporting compress, wound around the body, if possible up to the lower part of the abdomen decreases the accumulation of blood in the inclined parts.

2. The importance of regular and progressive physical activity.

3. A normal saline regime or with an increased sodium charge unless there is contra-indication (for example, cardiac insufficiency).

4. Medications.

There is little work available showing the effectiveness of numerous medications proposed, for a whole series of patients suffering from organic orthostatic hypotension. Most often the effect varies from one patient to another perhaps due to the poor understanding of etiological mechanisms in each particular case. Finally, the most effective medications are often those which are the most inconvenient.

- The sympathicomimetic (ephedrine, ethylephrine, norfenephrine, heptaminol ...) are effective by peripheral arteriole vasoconstriction (alpha affect). They also increase cardiac frequency and mycardium excitability (beta affect). They present risk of hypertensive pressure.

- Dihydroergotamine is a vasoconstrictor derived from ergot of rye in which the effects of vein constrictors seem to be better than other sympathicomimetics.

- Most recent publications show the inhibiting effect of

prostaglandines such as indomethacine which blocks the depressant effect of endogenic prostaglandines [6].

- The mineralocorticoids (fludrocortisone) increase the volume circulating and perhaps increase sensitivity to catecholamines. In general, they are effective but must be administered with care in patients when there is a risk of cardiac decompensation.

- Finally, one describes the benefit of administering medications /4 such as thyramine, the MAO inhibitors alone or in association with levodopa or even vasopressine. These are medications in which the secondary effects are not negligible and in which indication must be carefully weighed.

AFFECTIONS AND MEDICATIONS CAN INVOLVE ORTHOSTATIC HYPOTENSION. LOCALIZATION OF DAMAGE.

Afferent Tracts IX and X N.C.	Central Cerebral trunk	Efferent Tracts Medulla	Sympathetic chains + postganglion nerves
Tabes dorsalis Sugar diabetes Holmes-Adie syndrome B12 deficiency Ethylism	Familiale dysautonomy Cerebro-vascular affections Posterior cavity tumors Parkinson Plaque sclerosis Shy-Drager Medications: antihypertension tranquilizers, sedatives, hypnotic agents, antidepressants, phenothiazines, levodopa, etc.	Traumatism Tranverse myelitis Syringomyelia Intramedullary tumors Extramedullary tumors Degeneration of the intermediolateral collumn Amyotrophic lateral sclerosis Plaque sclerosis	Polyneuropathic polyradiculoneuritis: - diabetes - ethyl - paraneoplasia - uremia - porphyria - amyloidosis

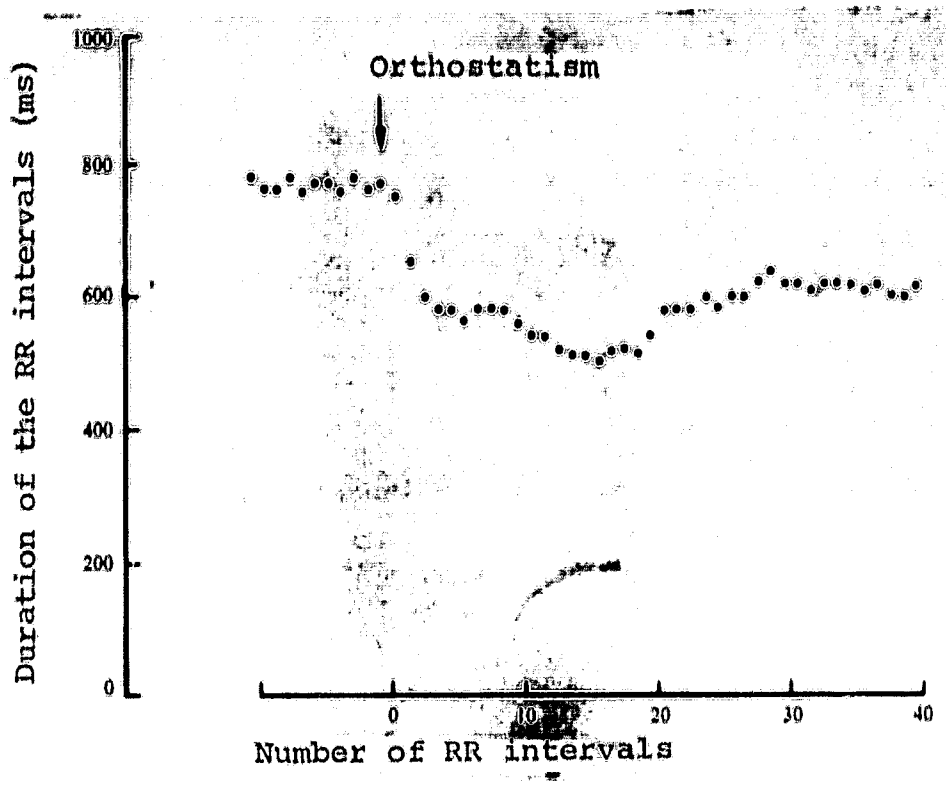

Figure 1. Development of cardiac frequency according to Ewing during an orthstatism test (healthy subject). One notes a tachycardia at the 15th beat and a bradycardia relative to the 30th.

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